Toxicology and Potential Health Risk of Chemicals that May Be Encountered by Workers Using Forest Vegetation Management Options

PART I: RISK TO WORKERS ASSOCIATED WITH EXPOSURE TO EMISSIONS FROM POWER SAWS

Forest Practices Branch
BC Ministry of Forests

Title Number 3

105-05647/

Abstract

Use of chain saws and brush saws in vegetation management under dense brush situations are expected to result in significant exposure of workers to exhaust fumes. Among the substances of concern in two-stroke engine exhaust are benzene and 1,3-butadiene, which are confirmed human carcinogens, and benzo(a)pyrene (BaP) a suspect human carcinogen. The exhaust contains carbon monoxide which interferes with oxygen transport to the cells of the body. Formaldehyde, acrolein and nitrogen oxides in exhaust are all irritants and may exert other systemic effects. Numerous other compounds are passed through in unburned gasoline, as combustion products or are formed by reactions in the atmosphere, but there is insufficient information to evaluate them.

Apparently, there are no studies of worker exposure to engine exhaust in brush management. However, data on exposure of loggers using chain saws provides information that can be used in preliminary estimates of the amount of these toxic substances in the breathing zone of brush control workers. Lifetime added cancer risk from benzene and 1,3- butadiene exposure associated with a typical employment history is estimated to be on the order of one to three chances in 1000 over the normal background of cancer in the human population. Risk associated with exposure to polyaromatic hydrocarbons, many of which are carcinogenic, or neurotoxic hydrocarbons cannot be assessed quantitatively at this time. Carbon monoxide exposure in dense brush is expected to be high enough to produce central nervous impairment, and increased risk of injury. Formaldehyde and acrolein are strong irritants and contribute to upper respiratory discomfort.

A program of exposure measurement is recommended in order to devise mitigation measures and to properly inform workers and policy makers of the potential risks in this kind of work.

National Library of Canada Cataloguing in Publication Data

Dost, Frank N.

Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part I, Risk to workers associated with exposure to emissions from power saws

Author: Frank N. Dost. Cf. Acknowledgements.
"Title number 3."
Includes bibliographical references: p.
ISBN 0-7726-4982-0

1. Two-stroke cycle engines – Exhaust gas – Health aspects. 2. Chain saws – Health aspects. 3. Weeds – Control – Health aspects. 4. Foresters – Health risk assessment. I. British Columbia. Forest Practices Branch. II. Title.

RC965.F59D67 2003 615.9'1 C2003-960120-X

DISCLAIMER: This publication is based on research information available as of the date this publication was written. The author and the project co-ordinator or others involved with the production of this paper have made reasonable efforts to avoid any errors. The publication is being made available as one source information that may be used together with such other sources of information, as one considers reasonable.

The use of product, trade or manufacturer's names in this paper does not constitute an official endorsement or approval by B.C. Ministry of Forests and its staff, or the author of any product or service to the exclusion of any others that may also be suitable.

Acknowledgements

This paper was prepared under contract for Forest Practices Branch, British Columbia Ministry of Forests, Victoria, B.C. by Dr. Frank N. Dost, DVM. Dr. Dost is a Fellow of the Academy of Toxicological Sciences, Emeritus Professor of Agricultural Chemistry and Forest Toxicology (Oregon State University, Corvallis), and Affiliate Professor in the Department of Environmental Health (University of Washington, Seattle). The preparation and review of the paper was coordinated by Dr. Jacob O. Boateng, (B.S.F., PhD), Provincial Vegetation Management Specialist, B.C. Ministry of Forests, Victoria.

The assistance of Shona Kelly (formerly Researcher, Health Care and Epidemiology, University of B.C., Vancouver and currently the Lecturer in Social Epidemiology at the University of Nottingham Medical School, Division of Epidemiology and Community Health [U.K.]), Dr. Ray Copes, Medical Specialist (B.C. Ministry of Health, Victoria and Medical Director, Environmental Health, B.C. Centre for Disease Control, Vancouver), Dr. Colin MacKay (Community Medicine Programme, University of B.C.), Vancouver, and B.C. Ministry of Forests staff in the review of this paper is acknowledged.

Funding for this paper was provided by the B.C. Ministry of Forests and Forest Renewal British Columbia (FRBC). Funding assistance by Forest Renewal B.C. does not imply endorsement of any statement or information contained in this publication.

Foreword

Vegetation management is an important reforestation activity for controlling competing vegetation or brush encroachment of young tree seedlings. The activity is necessary to get tree seedlings to free-growing status in most new forest sites established in areas that have been harvested or denuded by wildfire, insects and disease.

There are a number of options for managing forest vegetation. The treatment options include prescribed fire, herbicides, manual removal with hand and power tools (e.g., girdling and slashing tools, chain saws and brush saws), placement of mulch mats, mechanical techniques with heavy machinery, and biological methods. The use of livestock (e.g., sheep) is currently the common biological control technique employed in reforestation areas in British Columbia. Biological methods with insects or specific pathogens is used on forest rangelands for noxious weed control but not commonly used for vegetation control in young forest stands.

The selection of a treatment option involves a decision-making process based on integrated vegetation management concepts that include evaluation of the need for treatment, consideration of all the approved treatment methods and choosing the most appropriate treatment method, monitoring and evaluation. Factors considered in selecting a particular method are the ability of the method to meet the required reforestation objectives, the impact of the treatment at the specific site on human safety and the environment (e.g., recreational resources, fish and wildlife and their habitat, range resources and water supply), as well as the economics of the treatment.

This publication is one of a series of papers that evaluates the potential health effects on forest workers using the commonly employed methods of vegetation control. Other papers in the series are listed at the end of this paper. The emphasis is on risks associated with exposure to chemicals during the use of two most important methods for controlling competing vegetation in regenerated (natural or planted) forest areas. These methods are the use of herbicides and manual removal or control with handheld-motorized (power) equipment.

The herbicides discussed are those that have been commonly used in forestry in Canada. The database on health effects of herbicides is extensive and permits reliable estimates of risk. For components of chain saw exhaust and fuels, there is also voluminous background of toxicological information, but exposure data in forestry is limited. Nonetheless, there is enough information to develop preliminary assessments of potential health effects. While there appears to be a high incidence of physical injury associated with manual methods of brush control, there is virtually no validated data on which to base estimates of risk. The existing data are those of workers compensation boards and insurance companies but such data are generally difficult to obtain or are not specifically enough to characterize the kind of activity that leads to injury.

The information in these reports should provide the basis for important decisions about the way vegetation management in forestry should be carried out, and the use of some forestry activities as a source of assisted employment.

Contents

Abstract ii
Acknowledgements iii
Foreword iv
Introduction 1
Emissions from Two-stroke Engines and the Nature of Their Health Effects 1
Exposure to Exhaust from Power Saws 4
Estimation of Non-cancer or Systemic Risks 6
    Carbon monoxide (CO) 6
    Aldehydes, particularly formaldehyde and acrolein 7
Estimation of Cancer Risks 7
    Benzene 9
    1,3-butadiene 10
    Benzo(a)pyrene (BaP) and other PAH 11
Conclusions 11
Recommendations 12
References 13
Glossary 15
Titles in this Series 18

Introduction

The use of manual labour in forest vegetation management is perceived by some groups as a "safe" alternative to the use of herbicides, and is also seen to be a means of combating unemployment. However, every method of vegetation management carries with it some level of health risk to workers. For any operational objective the worker and public risks associated with each method of meeting the objective must be evaluated. The various adverse health effects of chain saw use and the potential interactions among them demand very careful attention when policies for vegetation management are developed. This report examines worker risks associated with exposure to exhaust emissions from power saws used in forest vegetation management. The well-studied deleterious effects of vibration and noise are not considered in this report, nor are physical injuries.

The emissions from two-stroke hand-held engines contain a large number of toxic compounds that are potential health hazards to humans. Some components are potent mutagens and are considered to be known human carcinogens. Others are respiratory irritants and central nervous system depressants.

Direct measurement of exhaust products in the breathing zone of brush management workers has not been done, but there is information on exposure of timber fallers, buckers and thinners in the field (Hagberg et al, 1985; van Netten et al, 1987; Nilsson et al, 1987a; Bünger et al, 1997). There is a wealth of anecdotal comment on headache, nausea, coughing and other symptoms in workers with chain saws, which is supported by more systematic worker surveys.

Brush management with power tools takes place in a variety of settings that have considerable influence on worker exposure to exhaust. In a relatively open environment with sufficient air movement, atmospheric concentrations of toxic exhaust products may be low enough to represent little or no health impact. On the other hand, work in deep brush and quiet air is likely to result in exhaust concentrations that not only directly impair health, but also cause central nervous effects that may result in lowered competence and consequent physical injury.

This report suggests that exposure to exhaust fumes confer potentially serious health risks, including increased risk of cancer, on workers who are doing brush control with power saws. It further recommends that a thorough study of exposure and physiological effects be carried out to protect the multitude of people who work with gasoline-powered equipment.

Emissions from Two-stroke Engines and the Nature of Their Health Effects

Levin et al (1984) and Nilsson et al (1987) have measured components of chain saw exhaust. A portion of their data is shown in Table 1. The hydrocarbon pattern of the exhaust was very similar to that of the gasoline used, leading to the conclusion that in the case of the engines studied, most of these components arose directly from the fuel.

Table 1. Concentration of chain saw exhaust components, at the saw (from Levin et al, 1984 and Nilsson et al 1987a)

Substance	Concentration (mg/m^3)	rel. std. dev. (%)
total hydrocarbons[a]	33,000	23
benzene	1400	16
total aldehydes	330	20
formaldehyde	120	16
naphthalene	14	84
benzo(a)pyrene	<0.005	
total PAH[b]	75	79
carbon monoxide	66,000	32
nitric oxide (NO)	45	48
other nitrogen oxides (NO_x)	50	55

[a] Total hydrocarbons include benzene, PAH, and other straight and branched chain compounds.
[b] Polyaromatic hydrocarbons, including naphthalene and benzo(a)pyrene.

Oestermark and Petersson (1992) studied vapour emission in refueling with conventional gasoline and a benzene-free alkylated gasoline that is marketed in Sweden for use in two-cycle engines. Benzene levels in alkylated gasoline fumes were reduced to levels approaching zero. The same authors (1993) analyzed exhaust composition from a two-cycle moped using alkylated and conventional gasoline. The engine is comparable to that of a large chain saw. Benzene levels in the exhaust from alkylated gasoline were about 50 fold less than in exhaust from conventional fuel. Methyl- and ethylbenzenes are significant toxicants, and are the source of much of the benzene in exhaust; they are almost 500 times more plentiful in conventional gasoline than in the reformulated product. Hexane, an important neurotoxic substance, was reduced 15 fold.

1,3-Butadiene emission is similar in both systems. Concentrations of the C2 through C5 alkenes (small hydrocarbons with one or more double bonds) are roughly similar, but hexene (C6) from alkylated fuel is reduced. All of the alkenes, which are roughly similar in the two fuels, have potential for genetic toxicity, particularly ethene. Most alkanes (saturated; no double bonds) are increased, because the objective of the reformulated fuel is a predominately branched, saturated chain product.

The importance of the quality of the fuel is demonstrated by the fact that about 30% of the fuel of a typical chain saw engine emerges unburned in the exhaust. (Nilsson et al, 1987) This loss by "flame quenching" is inherent as a function of the higher surface/volume ratio of smaller combustion chambers. As the ignition zone of the fuel/air mixture approaches the relatively cooler walls of the chamber, burning slows and the unburned mixture moves out with the exhaust (USEPA, 1994b). Also, a 2-stroke engine uses the incoming fuel charge to expel exhaust gases from the previous combustion event, inevitably blowing out some of the fuel (USEPA, 1999). These losses account for most of the benzene in the exhaust when conventional fuels are used.

Several of the substances or groups discussed above are known to be carcinogenic. Benzene is recognized as a known human carcinogen. Other than asbestos, benzene may be the most extensively studied of the human carcinogens because of wide use in industry. A decade ago, according to United States Environmental Protection Agency (USEPA, 1994) about 1.5% of typical gasoline was benzene, although content in some gasolines was probably higher. Present concentrations are about 1%.

1,3-butadiene is a combustion product present in two-stroke engine exhaust (Oestermark and Petersson, 1993; USEPA 1994), and is now identified by regulatory agencies as a known human carcinogen (USEPA, 1998).

Numerous polycyclic aromatic hydrocarbons (PAH) are found in two-stroke (and 4-stroke) exhaust, and many are animal carcinogens (IARC, 1983; ATSDR, 1993). Among these, benzo(a)pyrene is identified specifically because it is among the most potent of the PAH and is classified as a probable human carcinogen (IARC, 1987).

Polycyclic aromatic hydrocarbons may react with other combustion products or air contaminants to produce derivatives that are also of toxicological concern, although they have not been measured in exhaust from two-stroke engines. Probably of greatest importance are the products of reaction of PAH and nitrogen dioxide, also produced in combustion. Among these the nitropyrenes (nitrated four ring PAH) are described by Mermelstein et al (1981) as having "extraordinary" mutagenicity in bacterial systems, and as being the "most mutagenic chemicals reported in the literature." Tokiwa et al (1987), reviewing the mutagenicity and carcinogenicity of these products, use similar language. In spite of the extensive mutagenicity data and evidence of carcinogenicity of several nitro-PAH, the human health significance of the group is unclear (Tokiwa et al, 1987; Gibson, 1983). The importance of nitro-PAH cannot be appraised because of the absence of data.

The aldehydes in exhaust, including formaldehyde, are important because they are irritants. Formaldehyde, acrolein and nitrogen oxides probably account for most of the irritant quality of exhaust gases. The nitrogen oxides are also reactive with other constituents, resulting directly or indirectly in formation of PAH derivatives, ozone and other substances.

Carbon monoxide combines with haemoglobin to form a relatively stable compound, carboxyhemoglobin (COHb), which is unable to transport oxygen from the lungs to the cells and also inhibits normal haemoglobin from releasing oxygen at the cells where it is needed. If the intake of carbon monoxide is sufficient, oxygen lack at the cellular level will cause cardiovascular effects, weakness and central nervous depression. Physical and mental competence may be impaired sufficiently to result in injury. This factor may be a reason why a disproportionate fraction of chain saw-related injuries in New Zealand forest operations occur before midmorning, at a time of inversions and still air (personal communication, Richard Parker, Logging Industry Research Organization, Rotorua, New Zealand. 1995).

Small amounts of COHb are produced normally in the body; levels in unexposed individuals are on the order of 0.5% of total haemoglobin. Concentrations are somewhat higher in areas of incidental low-level air contamination as in cities. Smokers usually have levels of about 5%, although some heavy smokers may have as much as 15% of haemoglobin inactivated in this way.

The hydrocarbons collectively are direct central nervous system depressants, and may be responsible for the nausea reported by fallers and other users of chain saws.

Annual use of a single chain saw is roughly estimated by USEPA (1991) to produce approximately the same output of volatile organic compounds (VOC) as 9000 miles of driving passenger cars with current technology of that time. Elsewhere in the same document, an hour of use of a new chain saw is estimated to produce volatile organic emissions equivalent to those of 200 miles of driving a typical in-use automobile. The relation between these estimates is unclear (VOC include all of the hydrocarbons discussed above).

The term "in use" is not specified to mean the same as "current technology." If the two are the same, it appears that USEPA assumes only 45 hours of use annually for chain saws. The exposure estimate for use in brushing, discussed later in this report, assumes 20 hours a week for 20 weeks each year, or 400 hours. Other industrial uses should also be higher than 45 hours per year. In any case the point is clearly made that emissions from chain saw engines are significant.

Comparison of the USEPA emission data to those of Nilsson et al (1987) and Levin et al (1984) is difficult because the latter reports discuss emission in terms of milligrams of component per cubic metre of exhaust, and do not mention the volume of combustion gas produced. Oestermark and Petersson (1993) discuss the findings in terms of percent by weight of the exhaust output. The USEPA reports discuss the emissions in terms of grams of component per unit of power per hour. (hp-hr or kWh) Proportions were somewhat similar in the two reports, however.

Measurements of two of the most significant carcinogens were presented by USEPA (1994). A 3 hp (2.2kW) chain saw was found to emit about 10 grams benzene/hp-hr and a 6 hp saw produced 6.8 grams; the 6 hp saw produced 2 grams of 1,3-butadiene/hp-hr. There is not likely to be strict proportionality between benzene emissions and those of 1,3-butadiene at all horsepowers, because much of the benzene is carried through with unburned gasoline, while 1,3-butadiene is almost entirely a combustion product. In estimating exposure, however, the ratio will be considered constant.

Exposure to Exhaust from Power Saws

Once the toxicology of a chemical is known, the essential questions are exposure (the amount contacted) and dose (the amount absorbed). When toxicity and dose are known, an assessment of health risk is possible. Ideally, risk estimates should depend on the amount of the chemical that reaches the most sensitive target organ and its cells. As yet there is rarely sufficiently detailed information on distribution of the chemical or its reactive derivatives within the body to judge dosage at the cellular level.

Irritants, such as the aldehydes, are relatively reactive and affect the nose and throat rather than the lungs. Other components that are less reactive are of greater concern because they are more likely to reach the deeper portions of the lung, where they may be absorbed or retained without moving out in expired air. Most cancer-causing chemicals (including those found in exhaust) are of relatively low initial toxicity until converted to more reactive substances in the liver and other organs. Formation of these "reactive intermediates" is actually a step in detoxication, but becomes very important toxicologically at high doses of the compound.

The amount of material that actually reaches the respiratory tract depends on lung ventilation rate, which is well established for various rates of work. For the purpose of this assessment, the maximum breathing zone concentrations reported by Nilsson et al (1987) will be considered as the levels expected in still air. Because Nilsson et al did not measure 1,3-butadiene, the ratio between benzene and butadiene reported in USEPA (1994) will serve. A typical lung ventilation rate during hard physical work is about 2.5 m^3/hour. Hagberg et al (1985) measured pulmonary ventilation at 41 litres/ minute (2.46 m^3/hour) during felling of small trees. Bernstein (1978) estimated actual time on the saw in one brushing operation to be about 50% of the workday; it is assumed that a worker will be exposed for four hours a day. A four-hour stint would result in about 10 m^3 of air entering the lungs.

There have been no direct measurements of exposure to chain saw exhaust during brushing operations. Two papers already mentioned discuss exposure during logging (Nilsson et al 1987 and van Netten et al (1987) provide most of the information used to make preliminary estimates of exposure during brush removal. Bünger et al (1997) measured breathing zone carbon monoxide (CO) levels and the resulting COHb in loggers and thinners. Density of brush and more actual time on the saw are likely to result in much greater exposures during brushing operations, but until direct measurements are made, the data for loggers must be used. Reinhardt et al (T.E. Reinhardt, Personal Communication, 1995) measured several volatile compounds in the breathing zone of firefighters who were working wild fires, and found much higher benzene intakes when chain saws were being used.

Nilsson et al (1987) measured breathing zone concentrations of several components during logging in deep snow in a "sparse" pine forest and in a "thick" forest under snow free conditions. A portion of their data and the ranges are shown in Table 2. The wide variation seen argues strenuously for specific studies of exposure during brushing operations, under conditions expected in British Columbia forests.

Air movement was found to vary up to almost 3 m/sec (11 km/hr). The wind variation accounts for much of the wide variation in measured concentrations at the breathing zone samplers. In the absence of other information it may be assumed that still air would result in the highest breathing zone concentrations. Nilsson et al (1987) comment that their survey of operators showed that the worst subjective symptoms of exposure were associated with thick forest, calm weather and deep snow.

It is very clear from all of the exposure studies that the variety of work conditions typical of vegetation management with power tools can result in exposures that may be injurious

Table 2. Breathing zone concentrations of selected chain saw exhaust components. (Adapted from Nilsson et al, 1987a)

	Snow free[a]		With snow[a]	
Substance	Range, mg/m^3	TWA	Range, mg/m^3	TWA
total HC[b]	7–40	15	3–74	19
benzene	0.3–1.8	0.7	0.1–2.4	0.6
formaldehyde	0.04–0.2	0.08	0.02–0.1	0.08
PAH[c]	0.01–0.04	0.02	0.02–0.04	0.03
CO[d]	24–44	34	10–23	20

[a] Temp. range snow-free –3°–+8°C; with snow –16°–+1°.

[b] Hydrocarbons

[c] Polycyclic aromatic hydrocarbons.

[d] Carbon monoxide

Tetraethyl lead and dibromomethane were also measured but are not shown because leaded gasoline is not sold in British Columbia.

For this analysis, exposure to exhaust is to be considered in two modes, the maximum breathing zone concentration observed in the Swedish studies, and the time-weighted average (TWA) calculated in that work. The maximum concentration of benzene in the breathing zone measured by Nilsson et al (1987) was 2.4 mg/m^3 and the TWA over a seven-hour workday was 0.6 mg/m^3. Until more specific data can be obtained, these levels may be taken as an estimate of the expected range of exposure during work in deep brush with little air movement, and can serve as an index of other exhaust components.

The current exposure limit recommendation of the Workers' Compensation Board (WCB) of British Columbia for benzene is 2.5 ppm (8 mg/m^3) for 15 minute exposure and 0.5 ppm (1.6 mg/m^3) for eight hours.

The output of 1,3-butadiene in exhaust of a six-hp chain saw, according to USEPA (1994), is 12.2 grams per hour, compared to 40.8 grams of benzene. While Nilsson et al (1987) did not measure 1,3-butadiene, it may be assumed that the proportions were similar to those presented by USEPA. Because much of the benzene is carried through the engine in unburned gasoline and 1,3-butadiene is almost entirely a combustion product, the ratio may change with engines of different displacement.

At the higher benzene concentration of 2.4 mg/m^3, simultaneous 1,3-butadiene concentration would be 0.72 mg/m^3. WCB lists an eight-hour exposure limit of 4.4 mg/m^3. The basis for the published exposure limits is not clear. Presumably they are intended to avoid undefined systemic, reversible effects, because long term exposure at these levels would be associated with unacceptable cancer risks. Both substances affect blood forming tissues; for benzene the carcinogenic and non-carcinogenic effects are closely linked and probably not separable for practical purposes. 1,3-butadiene effects on bone marrow are unlikely at ambient or occupational exposures.

Exposure to benzo(a)pyrene (BaP) is difficult to estimate because it represents a very small fraction of total PAH and measurement is difficult. The studies of Nilsson et al (1987) suggested that BaP was less than the detection limit of 0.005 mg/cubic metre, which was 1/15,000 the total PAH observed in emissions, at the saw. PAH in exhaust was stated to arise almost entirely from unburned gasoline, based on comparisons of GC-MS analyses.

These findings were consistent with earlier work by Levin et al (1984), of the same group, indicating ratios of BaP/total PAH ranging from 1/8,000 to 1/15,000. However, this low output of PAH is quite different from those reported for other kinds of engines. Grimmer et al (1977, as quoted in IARC, 1983) found a ratio of BaP to total PAH of roughly 1/100 in emissions from air cooled four-stroke gasoline automobile engines of unspecified size, without emission control. While the respective engines are not similar, particularly in the use of the fuel lubricant mixture in two-stroke engines, it is difficult to reconcile a 150-fold difference.

The International Agency for Research on Cancer (IARC) comments on this problem. Discussing engine emissions: "Although there are many other such reports in the literature, they cannot be compared with one another

owing primarily to the use of different methods of PAH collection and differences in the reporting of unit measures of isolated amounts (e.g., microgram/litre fuel, microgram/travel distance, microgram/exhaust volume or microgram/gram particulate matter)."

van Netten et al (1987) monitored blood levels of COHb in and correlated this information with breathing zone measurements of carbon monoxide, lead and nitrogen oxides. Breathing zone concentrations of carbon monoxide were in the ranges observed by Nilsson et al (1987), with levels of 15-55 mg/m^3. These resulted in COHb levels between 1.5 and 3.4% of saturation. Bünger et al (1997) recorded carbon monoxide levels exceeding 500 ppm (780 mg/m^3) in some cases while limbing felled trees, which would be similar to working in brush. COHb levels of 5-6% were not uncommon.

Estimation of Non-cancer or Systemic Risks

For brushing operations, it is assumed that a typical work history consists of five four-hour days per week actually on the saw, 20 weeks per year, for five years but cumulation of exposure is short term. Systemic or non-cancer effects are reversible unless massive damage occurs, and when effects of low doses are either repaired or adapted rapidly enough, no effects may be seen even over long periods of exposure. Also, it is likely that at very low doses concentration of the chemical at potentially sensitive sites within the body is so low that no interaction can take place. When effects are only slowly reversible, as in the case of carbon monoxide intoxication, a 15-minute exposure and a one-hour exposure may have quite different outcomes because the impact accumulates over the longer period.

The non-carcinogenic compounds of greatest concern are carbon monoxide and the aldehydes, particularly acrolein and formaldehyde. While formaldehyde is an animal carcinogen, it is likely that it has a threshold, and impact in this area is probably very small relative to that of other exhaust gases. The impact of neurotoxic hydrocarbons, either simple depressants or the more destructive hexane, cannot be assessed in this report because of the lack of exposure data. Whether the variety of other hydrocarbons in exhaust represent only a nuisance or are toxicologically significant also cannot be evaluated at present. The nitrogen and sulphur oxides may be significant as irritants, but it is likely that their primary importance in chain saw exhaust lies in reactions with complex hydrocarbons, resulting in genetically active substances. Evaluation of these possible events is also not possible at present.

Carbon monoxide (CO)

Effects of various levels of CO exposure have been extensively studied in humans and have been reviewed by Amdur (1991) and by ACGIH (1991). In normal healthy unexposed individuals, carboxyhemoglobin (COHb) concentration varies around 0.5%, primarily as a result of normal breakdown of haemoglobin. General population levels are somewhat higher due to environmental exposure, particularly in urban areas. A COHb level of 2.5%, which can be achieved through exposure to about 65 mg CO/m^3 for 90 minutes, has been reported to interfere with time discrimination. A level of 5% COHb can be reached in an atmosphere containing 40-50 mg CO/m^3 over four hours, and has been reported to cause impairment of various motor functions. The significance of such effects in a worker using dangerous tools is obvious. Although workers in brush control are not likely to have coronary artery disease, modest exposure to CO is acutely threatening to individuals with such a condition. If exposure to CO in exhaust is added to levels already acquired by a smoker, the degree of response will also be additive. Some smokers may carry COHb levels as high as 15%. Whether an additional insult can be tolerated is not clear. Sustained CO exposure has been observed to result in increased red blood cell concentration,

presumably as adaptation to lowered oxygen carrying capacity.

The maximum COHb level measured in timber fallers by van Netten et al (1987) was 3%, resulting from an exposure to a time-weighted average (TWA) air concentration of 36 ppm (41 mg/m^3). Average concentration during actual use of the saw was 55 ppm (63 mg/m^3). The nature of the work is such that fallers are exposed intermittently for relatively short periods. Nilsson et al measured a TWA of 34 mg/m^3 during snow-free conditions and 20 mg/m^3 with snow at the site. The highest breathing zone concentration was 44 mg/m^3, apparently in quiet air, during felling operations. Unfortunately, data on CO exposure was obtained from only one faller during four work periods in each of the two kinds of working conditions. Bünger et al (1997) found that COHb levels of 5-6% were not uncommon while limbing felled trees, which is an environment similar to heavy brush. They noted that workers were often in a crouched position, with the breathing zone close to the exhaust, a situation similar to that of brushing operations. Also, video recordings of the work showed that exhaust did not rise, but remained in the vicinity of the worker, unless carried by the wind. Bünger et al (1977) did not comment on evidence of acute health effects.

Carbon monoxide intoxication is reversible, but recovery is slow. The half-recovery time in a normal atmosphere is on the order of five hours. Therefore, even if exposures are interrupted throughout the day, the concentration of COHb may continue to increase stepwise because formation is faster than recovery. There will still be some residue the following day.

The upper ranges of breathing zone CO concentration observed by Nilsson et al (1987), Hagberg et al (1985), van Netten et al (1987) and Bünger et al (1997) are probably sufficient to raise blood COHb levels to 5% or more in four hours of working time during a day (Amdur, 1991).

It appears likely that workers using chain saws in brush when ventilation is poor are likely to absorb enough CO to impair performance and increase risk of physical injury. Because specific exposure data for this kind of work is lacking, a numerical estimate of risk is not possible. Whether CO is a contributor to the headache, nausea and other effects reported by the loggers surveyed by Nilsson et al (1987) and by Hagberg (1985) is arguable, but the overall effect of exhaust on these workers shows that exposure to all components is high. The apparent high probability of such effect dictates that measurements of CO exposure and COHb are urgently needed.

Aldehydes, particularly formaldehyde and acrolein

The data of Nilsson et al (1987) and Hagberg et al (1985) indicate that exposure to formaldehyde may reach 0.2 mg/m^3 (0.13 ppm). Although acrolein is somewhat more irritant than formaldehyde, its irritant mechanism is similar, and studies of rodents suggest that the two aldehydes act at a common site and compete with each other. Their effects together are therefore less than the sum of each alone. (Kane and Alarie, 1978)

On the basis of data summarized by Bernstein et al (1984) and Hagberg et al (1985) it may be concluded that concentrations of aldehydes in the breathing zone during brushing operations are within the range that can cause irritation and breathing difficulty. No quantitation can be applied in this case because the amounts brought into the respiratory tract are not known, and there is high variability in human responses.

Estimation of Cancer Risks

This section includes estimates of the risk of cancer associated with exposure to certain of the cancer-causing substances in the exhaust of chain saws as used in vegetation control.

The significance of the duration of respiratory exposure differs between carcinogens and non-

carcinogens. In both cases, exposure is assumed to represent dose, although some escape in expired air may occur, depending on the physical make-up of the breathing atmosphere. For substances that may be carcinogens, current regulatory risk models assume that probability of a carcinogenic effect is directly related to the cumulative total dose over a lifetime. In other words, a given dose will have the same effect whether acquired at a high rate over a short period or a low rate over a long term. The total dose acquired is converted to an average daily dose over a 70-year (25550-day) lifespan for cancer risk estimates.

Risk estimates are always expressed as added risk over the existing very high background; 30 percent or more of North Americans will have cancer during their lifetime. Incidence of some kinds of cancer is rising, others are becoming less frequent. Overall incidence on an age-corrected basis has not changed markedly over several decades. However, as other diseases are better controlled, life span is increasing and the population is becoming older, which raises the absolute background frequency of cancer.

Cancer risk assessment uses existing information to predict a future burden of cancer that some specified activity may add to the background incidence. The same idea may be stated as estimating the chance that an individual engaged in that activity will be affected, in addition to the one chance in three with which we are already burdened. The value of risk assessment in its present form is as much in comparison of chemicals in some standard manner as it is in prediction of specific future risk.

Estimation of cancer risk for regulatory purposes currently is based in part on several assumptions or generalizations. Perhaps the most important assumption is that information from animal studies is applicable in estimating human cancer risk. At present there is little argument that this is true, although there is debate over the degree of applicability. There are numerous substances for which there are substantial differences in sensitivity or dose response of specific organs among species.

A very important quantitative assumption is that a given intake of a chemical will carry the same risk whether acquired over a short time or over a lifetime. The validity of that idea has yet to be fully established, but as yet it is the most conservative assumption and is applied to most cancer risk estimation. The question is of particular interest here because there is evidence that as the time elapsed after industrial exposure to benzene increases, leukaemia risk lessens (Finkelstein, 2000).

At present, estimation of cancer risk is not able to fully utilize data on the way chemicals move in the body and the way they are changed chemically by normal metabolic processes. In the regulatory context, distinction is usually not made between chemicals (or their products) that act directly on genetic material (DNA), and chemicals that act indirectly through systemic effects.

These uncertainties aside, there is no question that carcinogens act in a dose related fashion. Just as for all other kinds of effects, as the intake of a chemical increases, the response increases. In the case of chemicals that cause cancer, the dose governs the frequency or probability of occurrence and in some cases the time required for tumors to develop.

Where epidemiological information on increases in human cancer incidence relative to environmental or occupational exposure is available it may also be used in finding a dose response relationship. However, there are few chemicals that have been reliably associated with increased human cancer; among those of concern here, both benzene and 1,3-butadiene are considered by USEPA to be known human carcinogens (USEPA 1997, 1998) on the basis of associations with increased human cancer incidence.

To deal with these various uncertainties a number of models for estimating risk have been proposed. The presumed most conservative

model for estimating risk in the regulatory context assumes a linear relationship between dose and effect from the high doses used in animal studies down to very low doses. A factor is included that corrects for size difference, which is only partly related to body weight. A statistical uncertainty factor is calculated to indicate the range within which the true dose-response slope must lie, and the upper bound or most conservative slope is used for estimation of human risk. It is evident that with this approach it is assumed that even the smallest imaginable dose carries a proportionate risk. The regulatory philosophy is that there is a low dose rate below which risk or probability of an effect is so low that it cannot be distinguished from zero.

A typical dose-response curve at higher levels, and the kind of dose-response slope developed for the very low doses in human exposures are shown in the discussion of Principles of Health Effects Evaluation. A given average daily dose of a chemical over a lifetime relates to the estimated probability that the dose will cause cancer in excess of the normal background incidence.

The standard dosage term is one milligram (mg) or one microgram (ug) of chemical per kilogram (kg) body weight per day. The potency of a carcinogen is usually described as an exponent, for example, 2×10^{-3} per (mg/kg/day). This means that a dose of one mg/kg/day over a lifetime would be predicted to carry a cancer risk of two chances in a thousand, over the background of more than 300 chances in a thousand. Because most exposures are not continuous over a lifetime, shorter-term exposures are totalled and averaged over a standard 70 year span. Such unit risk or potency figures are based on biological and statistical assumptions that are intended to maximize risk estimates.

For airborne carcinogens, the average daily dose is often expressed in terms of concentration in air. The potency term is then described as mgm^3 average concentration in breathing air over a lifetime.

Because there is insufficient information upon which to base a more complex model, this assessment will utilize an estimation of exposure for a five-year work history. The exposure estimate will be distributed over a 70-year lifetime for risk estimation. This will enable a preliminary judgement of the magnitude of cancer risk associated with exposure to chain-saw exhaust and can be used in decisions both for managing the risks and determining what further analytical studies must be done to assure safety in brush management.

Benzene

Benzene is considered to be a confirmed human carcinogen, causing leukaemia (USEPA, 1994b; 1997). There is debate about its association with increased incidence of non-Hodgkins lymphoma (Wong and Raabe, 2000a; 2000b; Goldstein and Shalat, 2000a), and with multiple myeloma (Bergsagel et al, 2000; Goldstein and Shalat, 2000b). Thorough reviews have been prepared by Duarte-Davidson (2001) and Hayes et al (2001)

Based on the work of Nilsson et al (1987) the maximum case total exposure would be ($2.4\ mg/m^3$) × ($2.5\ m^3$/hour) respiration × 2000 working hours = 12,000 mg, assuming efficient absorption. At the TWA, total exposure is 3,000 mg. For a 70-kg worker the respective total doses are 171 and 43 mg/kg. Averaged over a 70-year (25550-day) lifetime, the dose rates are approximately 0.007 and 0.0017 mg/kg/day.

Unit risk for a volatile carcinogen may be expressed in terms of either air concentration or dosage. USEPA (1994b, 1999) notes a range of potencies based on exposure to benzene in air over a lifetime ranging from 8.3×10^{-6} to 5.2×10^{-5} per ug/m^3. In terms of dosage, these figures correspond to unit risks of 2.9×10^{-2} and 1.81×10^{-1} per (mg/kg/day) (USEPA, 1993). These are the estimated 95% upper bound values; statistically the actual potencies are 95% certain to fall somewhere below these levels. At the lower level an average dose of 0.001 mg/kg/day

over a 70-year lifetime would confer a maximum theoretical added risk of leukaemia, over background of 2.9 chances in 100,000 lifetimes. At the higher level the maximum theoretical added risk would be 1.8 chances in 10,000. Actual added risk could be as low as zero.

For the work period specified above, at the lower unit risk figure, the estimated dose rate of 0.007 mg benzene/kg/day would confer an added risk over background of 2×10^{-4}, or 2 chances in 10,000 lifetimes. [2.9×10^{-2} per (mg/kg/day) $\times$ 0.007 mg/kg/day = 2×10^{-4}] The daily dose based on the time-weighted average exposure, 0.0017 mg/kg, confers an added risk of about 5.2 chances in 100,000.

If the higher estimate of risk turns out to be correct, the theoretical maximum risks associated with these exposures will be about 1.3 chances in 1,000 and 3 chances in 10,000.

The estimates of risk that may be imposed on a worker doing brush control work with a chain saw under the estimates just discussed cover about two orders of magnitude, which is not a wide range for such predictions. Even though there are significant uncertainties, it is quite clear that there should be concern about carcinogenic impact on these workers. Some regulatory schemes consider estimated risks on the order of one in 10,000 or even higher ($>10^{-4}$) to be acceptable in the industrial context. It is difficult to rationalize that philosophy, even though the practical likelihood of any individual being affected in, say, a given 100-person work force is very low. At the same time, it is much higher than the range of estimated risk generally assumed to be virtually equal to zero, 10^{-6}-10^{-5}, which is generally applied in evaluating public health as distinguished from occupational health criteria.

To summarize, risk associated with estimated benzene exposure ranges between about 1.3 chances in 1,000 and 5.2 chances in 100,000, depending on the potency and exposure estimates. If the apparent short latency (period between initial exposure and appearance of clinical disease) for leukaemia caused by benzene is considered, the risk estimates may become much higher. Furthermore, it is evident that there are differences among humans in the metabolic processing of benzene which are almost certain to characterize more sensitive segments of the population.

1,3-butadiene

USEPA has recently classified 1,3-butadiene (BD) as a known human carcinogen and has developed a new carcinogenic potency estimate for average lifetime intake that is lower than the older estimate of potency for average lifetime total intake (USEPA, 1998). The new figures are based on incidence of leukaemia in studies of occupationally exposed industrial workers. Until recently, estimates of carcinogenic potency in humans were conservatively based on data from studies of mice, which are more sensitive than humans or rats. In addition, recent research indicates that metabolism of 1,3-butadiene to its DNA-reactive metabolites is much more extensive in mice than in rats or humans (Himmelstein et al, 1994; Medinsky et al, 1994). Various other cancers have also been found in animals exposed by inhalation of BD. The mutagenicity of BD is clearly established.

(There is some confusion among USEPA publications. A 1999 Hazard Summary 106-99-0, from the Office of Air Quality Planning and Standards, still refers to the older potency estimate and the older classification as a "probable" rather than "known" human carcinogen.)

The 1998 life-time risk estimate of 9×10^{-3} per ppm (3.7×10^{-3} per (mg/m^3) is considerably lower than the earlier estimate of 2.8×10^{-1} per (mg/m^3) (3.7 chances in 1,000 per mg/m^3) vs. 2.8 chances in 10 for lifetime average exposure). An air concentration of 3.7×10^{-3} per (mg/m^3) is equivalent to a dose rate of about 1.3×10^{-2} per (mg/kg/day).

The ratio of butadiene to benzene is taken to be 0.3; lifetime exposure is therefore 4000 mg. At 70-kg body weight, total dose per kg is 57 mg.

which averages to 0.0022 mg/kg/day over a lifetime at the higher exposure and 0.00054 mg/kg/day at the TWA.

The estimated added lifetime risk at the high exposure is (0.0022 mg/kg/day) × (1.3×10^{-2}/(mg/kg/day)) = 2.86×10^{-4}, which is about 3 chances per 100,000 lifetimes. At the TWA exposure added risk would be about 7.5 chances per million.

Benzo(a)pyrene (BaP) and other PAH

There is no question that BaP is a potent animal carcinogen, and numerous other PAH found in exhaust are also carcinogenic, a few of which may be as active as BaP. Of 33 PAH identified in gasoline engine exhaust by Grimmer (1977, as quoted in IARC (1983), 10 are considered by IARC to be carcinogenic, 5 show limited evidence of carcinogenicity, for 15 the data are inadequate to make a judgement. Only three are considered to be not carcinogenic.

There is little information about the amounts of PAH and related compounds in two-stroke engine exhaust, and no data on exposure during vegetation control activity. Measurements have apparently been published by only one group (Levin, et al, 1984; Nilsson et al, 1987) Their findings suggest that PAH concentrations in chain saw exhaust are very low, but because output from other kinds of gasoline engines are known to be much higher, the exposure question must be considered to be unanswered at present. If levels of PAH in two-stroke exhaust are actually as high as those from other engines, exposures may become significant.

Another concern is the potential for formation of carcinogenic derivatives of PAH either during combustion or in the exhaust mix as it emerges. An example is the reaction of nitrogen oxides with PAH. The potential for formation of nitro-PAH in chain saw exhaust seems substantial, but there are no data for this kind of equipment. Many members of this group are mutagenic and carcinogenic {see, for example, King (1988) and Tokiwa et al (1987)}, which is a warning that emission measurements for this family of compounds should also be obtained.

With such uncertainty an attempt at quantitative estimation of risk is futile. The information at hand does suggest that the PAH in exhaust may confer undesirable health impacts, which would add to those arising from other PAH sources, most notably from automobiles and other equipment, and from residential heating by wood.

Conclusions

Workers using chain saws (and brush saws) are exposed to benzene and 1,3-butadiene which are considered to be known human carcinogens. Very few chemicals are so classified. Polyaromatic hydrocarbons, several of which are suspect human carcinogens, are also in the exhaust mix as is carbon monoxide.

Available data suggest that exposures to these substances may reach unacceptable levels under some working conditions. The sum of excess cancer risks (without an estimate for PAH or other suspected genetic toxicants in exhaust) is estimated to be on the order of 10^{-3} to 10^{-5} (one chance in 1,000 to one chance in 100,000). As time-to tumour (latency) of benzene, and differences among individuals in metabolizing benzene are better defined, risk estimates may change.

Carbon monoxide and neurotoxic hydrocarbon exposures may be sufficient to cause systemic intoxication and impair work performance and safety.

It is imperative that studies be conducted in the field to measure worker exposure under the variety of conditions of manual brush management. Only with such measurements can it be determined whether such work is taking place under conditions of acceptable safety.

Recommendations

1. While the estimated significant cancer risks associated with exposure to benzene, 1,3-butadiene and BaP may not be taken as a signal for immediate operational disruption, they do demand immediate study of exposures (breathing zone atmospheres) under the various work conditions of manual brushing. An evaluation of the importance of other hydrocarbons to worker health and safety is also critical.
2. Breathing zone exposure measurements of CO should be coupled with sequential blood analyses of carboxyhaemoglobin (COHb) during and after work periods, with simultaneous analysis of physical performance to determine the true impact of carbon monoxide (CO) exposure in brushing operations.
3. Along with analytical work, a survey of subjective operator experience should be carried out. Other studies have indicated that loggers experience nonspecific intoxication leading to headache, nausea and respiratory irritation.
4. Mitigation measures should be explored, even if exposure information is not complete.
   a. Mitigation might take the form of working with power equipment only at some minimum on-site wind level, which will dilute the emissions and the risks.
   b. Engineering solutions to the exposure problems should be explored. For example, it may be possible to lead exhaust away from the operator in such a way that it has velocity enough to move some distance before diffusing. A typical saw muffler diffuses the flow of gases in every direction, at the saw. Exposure of other workers must be considered in such an effort.

      Efforts to design four-cycle engines and catalytic converters for hand-held applications are underway, with some limited introduction to the market already. In the case of chain saws, which often operate inverted, success does not seem likely. USEPA is proposing emissions regulations for non-road engines of all classes, and in co-operation with manufacturers is evaluating engineering changes that will achieve these goals. This type of research should be considered in this effort to devise new generation machines.
   c. Benzene and other highly toxic substances reach the breathing atmosphere as part of the unburned fuel. Steps to require reformulated gasoline, free of benzene and some other toxic components should be explored, drawing on the Swedish experience, where such requirements are in place.
   d. It is not likely that or respirators are a useful solution to exposure in this kind of work. Respirators require care, replacement of filters, and proper fit, and when not working properly can cause more harm than they prevent. Comfort and convenience are low, and vision is likely to be obstructed, which is safety impairment itself.
5. Continuing attention should be paid to advances in the field of risk assessment, for most effective use of any exposure data obtained.
6. If manual brush control with power tools is to be used as a means of reducing unemployment or as an entry into the forestry labour pool, the ethical and policy implications of the potential accompanying health risks must be given close attention. Such policy decisions are beyond the realm of scientific analysis, but must include an objective and thorough examination of the risks for each option. A requirement of informed consent of workers, with the attendant education and training efforts may be necessary. Such labour as manual brush control is taken for granted as "safe" by workers, employers, resource management agencies, and the public. This lack of understanding must be dealt with if future liability is to be avoided.

References

ATSDR (Agency for Toxic Substances and Disease Registry. (1993) Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs) Draft For Public Comment. US Public Health Service.

ACGIH (American Council of Government Industrial Hygienists) (1991) Documentation of Threshold Limit Values and Biological Exposure Indices. Sixth Edition.

Amdur, M. O. (1991) Air Pollutants. In "Toxicology, The Basic Science of Poisons", Eds. Amdur, M. O., Doull, J. and Klassen, C. D., pp 854-871. Pergamon Press, New York.

Bergsagel, D.E., Wong, O., Bergsagel, P.L., Alexanian, R.,Anderson, K.C., Kyle,R.A. and Raabe, G.K.(1999) Benzene and multiple myeloma: Appraisal of the scientific evidence. Blood 94:1174-1182.

Bernstein, A. (1978) Using a chainsaw to control brush. J. Forestry 76:474-475.

Bernstein, R.S., Stayner, L.T., Elliott, L.J., Kimbrough, R, Falk, H. and Blade, L. (1984) Inhalation exposure to formaldehyde: An overview of its toxicology, epidemiology, monitoring and control. Am. Ind. Hyg. Assoc. J. 45:778-785.

Bünger,J., Bombosch, F., Mesecke, U. and Hallier, E. (1997) Monitoring and analysis of occupational exposure to chain saw exhausts. Am. Ind. Hyg. Assoc. J. 58:747-751.

Duarte-Davidson, R., Courage, C., Rushton, L. and Levy, L. (2001) Benzene in the environment: An assessment of the potential risks to the health of the population. Occup. Envir. Med. 58:2-13.

Finkelstein, M.M. (2000) Leukemia after exposure to benzene: Temporal trends and implications for standards. Am. Jour. Indust. Med. 38:1-7.

Gibson, T. L. (1983) Sources of direct-acting nitroarene mutagens in airborne particulate matter. Mutation Res. 122:115-121.

Goldstein, B.D. and Shalat, S.L. (2000a) Letter: Non-Hodgkins lymphoma and exposure to benzene in petroleum workers. Jour. Occup. Envir. Med. 42:1133-1134. (Response: Wong and Raabe, Jour. Occup. Envir. Med. 42:1134-1136.)

Goldstein, B.D. and Shalat, S.L. (2000b) Letter: The causal relation between benzene exposure and multiple myeloma. Blood 95:1512-1513. (Response: Bergsagel et al, Blood 95:1513-1514.)

Hagberg, M., Kolmodin-Hedman, B., Lindahl, R., Nilsson, C-A. and Norstrom, A. (1985) Irritative complaints, carboxyhemoglobin increase and minor ventilatory changes due to exposure to chain-saw exhaust. Eur. J. Respir. Dis. 66:240-247.

Hayes, R.B., Songnian, Y., Dosemeci, M. and Linet, M. (2001) Benzene and lymphohematic malignancies in humans. Am. Jour. Indust. Med. 40:117-126.

Health Effects Institute (1999) A partnership to examine emerging health effects: EC/HEI Workshop on 1,3-Butadiene, Brussels, Belgium, convened in June, 1998.

Himmelstein, M.W., Turner, M.J., Asgharian, B. and Bond, J.A. (1994) Comparison of blood concentrations of 1,3-butadiene and butadiene epoxides in mice and rats exposed to 1,3-butadiene by inhalation. Carcinogenesis 15: 1479-1486.

IARC. International Agency for Research on Cancer. (1983) IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol 32, Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data. WHO, Lyon, France.

IARC. International Agency for Research on Cancer. (1987) IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Suppl. 7.

Kane, L.E. and Alarie, Y. (1978) Evaluation of sensory irritation from acrolein-formaldehyde mixtures. Am. Ind. Hyg. Assoc. J. 39:270-274.

King, C.M. (1988) Metabolism and biological effects of nitropyrene and related compounds. Health Effects Institute Research Report 16.

Levin, J-O., Nilsson, C-A. and Norstrom, A. (1984) Sampling and analysis of polycyclic aromatic hydrocarbons (PAH) from two-stroke chain saw engines. Chemosphere 13:427-435.

Medinsky, M.A., Leavens, T.L., Csanady, G.A., Gargas, M.L. and Bond, J.A. (1994) In vivo metabolism of butadiene by mice and rats: A comparison of physiological model predictions and experimental data. Carcinogenesis 15: 1329-1340.

Mermelstein, R., Kiriazades, D.K., Butler, M. McCoy, E.C. and Rosenkrantz, H.S. (1981) The extraordinary mutagenicity of nitro-pyrenes in bacteria. Mut. Res. 89:187-196.

Nilsson, C-A., Lindahl, R. and Norstrom, A. (1987) Occupational exposure to chain saw exhausts in logging operations. Am. Ind. Hyg. Assoc. J. 48:99-105.

Oestermark, U. and Petersson, G. (1992) Assessment of hydrocarbons in vapours of conventional and alkylate-based petrol. Chemosphere 25:763-768.

Oestermark, U. and Petersson, G. (1993) Volatile hydrocarbons in exhaust from alkylate-based petrol. Chemosphere 27:1719-1728.

Tokiwa, H., Nakagawa, R., Horikawa, K. and Ohkubo, A. (1987) The nature of the mutagenicity and carcinogenicity of nitrated, aromatic compounds in the environment. Envir. Health Persp. 73:191-199.

USEPA (Environmental Protection Agency) (1991) Nonroad Engine and Vehicle Emission Study--Report. Office of Air and Radiation, PB 92-126960.

USEPA (Environmental Protection Agency) (1993) Selecting Exposure Routes and Contaminants of Concern by Risk-Based Screening. EPA Region III, Hazardous Waste Management Division. EPA/903/R-93-001.

USEPA (Environmental Protection Agency) (1994) Draft Regulatory Impact Analysis and Regulatory Support Document. Control of Air Pollution; Emission Standards for New Non-road Engines At or Below 19 Kilowatts. Office of Mobile Sources, 2565 Plymouth Road, Ann Arbor, MI, USA 48105.

USEPA (Environmental Protection Agency) (1997) Carcinogenic Effects of Benzene: An Update. EPA/600/p-97/001A. Office of Research and Development.

USEPA (Environmental Protection Agency) (1998) Health Risk Assessment of 1,3-Butadiene. EPA/600/P-98/001A. Office of Research and Development.

USEPA (Environmental Protection Agency) (1999) Supplemental Draft Regulatory Impact Analysis. Phase 2 SNPRM: Emission Standards for New Non-road Engines At or Below 19 Kilowatts. Office or Air and Radiation, Washington, D.C.

van Netten, C., Brubaker, R.L., MacKenzie, C.J.G. and Godolphin, W.J. (1987) Blood lead and carboxyhemoglobin levels in chainsaw operators. Envir. Res. 43:244-250.

Wong, O. and Raabe, G.K. (2000) Non-Hodgkin's lymphoma and exposure to benzene in a multinational cohort of more than 308,000 petroleum workers, 1937 to 1996. Jour. Occup. Envir. Med. 42:554-568.

Glossary

Adjuvant – Any additive to a pesticide formulation that is not active itself, but is intended make the active ingredient work better.

Cancer – A malignant growth of potentially unlimited size that invades local tissues, and may spread to other parts of the body.

Carcinogen – A chemical capable of inducing cancer.

Carcinogenic – Capable of causing cancer

Contaminant – In a formulation, usually residues or impurities from the manufacturing process present in small quantities. Contaminants must be identified to the regulatory agency, which judges whether they are of concern.

Deoxyribonucleic Acid – See **DNA**

Detoxication (Detoxification) – The biochemical process of changing a chemical in the body to a less toxic form or to a form that can be more easily excreted.

Dose – The amount of a chemical that actually enters the body to be distributed to all of the organs and cells. Distribution to tissues and cells is selective, and depends on the nature of the chemical and characteristics of each kind of cell.

Dose-response relationship – The central idea in toxicology and in pharmacology (which is the science dealing with beneficial effects of therapeutic drugs). As the dose (or concentration) of a chemical increases, the effect increases, and as the dose is lowered, the effect becomes less. This response pattern applies to every interaction between a chemical and a biological system, whether human, fish, bacteria or any other kind of organism or tissue. The dose-response relationship is absolutely essential to judgement of the effect of any chemical.

DNA (Deoxyribonucleic Acid) – The genetic library in each cell that contains all of the instructions for building and operating the body. Each kind of cell contains all of the information for the whole body. Only the information needed for each kind of cell is used by that cell; the rest is repressed. Liver cells do not try to be muscles, and muscles do not try to become brain cells, but they contain all of the information.

Epidemiology – The scientific study of the cause, distribution, and control of epidemics or other disease in a region. In the context of these reports, epidemiology is the study of possible associations between environmental and occupational chemicals and occurrence of diseases. The term "associations" is used in its statistical sense, which means that the relationship cannot demonstrate cause and effect.

Exposure – Amount of a chemical that reaches a surface from which it might be absorbed. The dose is some fraction of the exposure. Exposure does not include material that is on nearby foliage or other surfaces. It is only the material that reaches the skin (by contact), respiratory tract (by inhalation) or digestive tract (by ingestion).

Formulation – A complete pesticide preparation as sold by a manufacturer for practical use. It includes the active ingredient and any necessary adjuvants and solvents. For use, it may or may not require further dilution or mixing with other substances. Formulation can also be defined as the process used by manufacturers in preparing a pesticide for practical use.

Half-life – The length of time required for disappearance of half of the material present in an organism or in environmental media. It is a more useful idea than "persistence" because it allows prediction of the time required to reach low target levels with out making measurements over exceedingly long periods. A better term is "Half-time," because the information only relates to a given location, and says nothing about the processes that deplete the chemical. If it evaporates or is carried away intact by water it may still exist in its original form. The term "half-life" originated with description of radioactive decay, in which elements become a totally different substance. The English language sometimes loses precision as it evolves.

Hazard – The kind of effect that a chemical can cause. Cancer, liver disease, skin irritation, reproductive problems, or some other more or less specific response that can be defined and measured. The term is also used non-specifically to signify any dangerous situation.

Herbicide – A chemical substance or cultured biological organism, used to kill or suppress the growth of plants.

Immune system – All of the structures and cells and their products that protect against infectious organisms and against cells of the body that have become altered in the very early development of cancer.

Irritation – A purely local or topical reaction which may include redness, blistering, swelling, burning or itching.

Lethal – Causing death.

LOAEL – Acronym for lowest-observed-adverse-effect level.

Lowest-observed-adverse-effect level (LOAEL) – The lowest measured amount of a chemical that produces significant increases in frequency or severity of adverse effects in exposed subjects. in the general sense it includes all biochemical, pathological, behavioral, reproductive, genetic and other measurable changes. the term may also be applied to any specific parameter under observation.

Malignant – Deadly or very injurious. As applied to cancer, invasive of local tissues and metastatic (migration of cancer cells to other tissues).

Margin of Safety (MOS) – The difference between the estimated dose of a pesticide and the NOAEL. A **MOS** of 100 (estimated dose 100 fold less than the NOAEL) is usually considered to assure that no adverse effects will occur.

Metabolism – the sum total of the biochemical reactions that a chemical undergoes in an organism. The processes include biochemical (enzymatic) reactions in the cells of the body that convert nutrients to energy and structural materials of the body; reactions that change wastes so they can be removed; and reactions that convert foreign substances, such as some pesticides to forms that can be excreted.

MOS – Acronym for margin of safety.

Mutagenic – Capable of producing genetic changes.

Mutagens – Chemicals that are able to induce gene or chromosome damage that is stable and survives cell division to reach the next generation of cells. See **mutation.**

Mutation – Genetic change in DNA of a cell that can be transmitted to the next generation of cells. If in sperm or egg cells, a mutation may be transmitted to offspring. If in somatic (body) cells such as liver, muscle or other organs, a mutation may pass to daughter cells in the organ. The change may have no effect on cell function or it may damage the cell, or even imaginably improve it.

NOAEL – Acronym for **no-observed-adverse-effect level.**

No-observed-adverse-effect level (NOAEL) – The dose rate or concentration at and below which no adverse effects can be detected. (See **threshold; SEE LOAEL)** If the estimated dose of a herbicide to a worker is very low compared to the **NOAEL** for the most sensitive effect found in the laboratory, no harmful effect is to be expected.

Persistence – The duration of measurable concentrations of a pesticide in soil, foliage or other media. (See Half-life.)

Pesticide – Any chemical (or biological product) intended to control or kill pests. Herbicides, insecticides, fungicides are all pesticides. The term is sometimes incorrectly used to mean only insecticide, for example "pesticides and herbicides."

Registration – The process by which government (e.g., Canadian federal government) authorities determine that a pesticide is suitable for use. Standards of public and worker safety, environmental impact, and usefulness must all be met.

Risk – The probability (likelihood) that some adverse or undesirable effect will take place in the future, as a result of some specified activity. Risk may relate to health, finances or any other kind of undesirable impact. Real risk may be so small that it cannot be distinguished from zero, or so great that it is a certainty. In the context of pesticides, risk is the probability that use of the pesticide will result in some specified harmful effect on workers or the public. Risk assessment is the process of estimating that probability.

Safety Factor – See **Margin of Safety.**

Teratogen – A chemical that can cause birth defects.

Teratogenic – Relating to or able to produce birth defects.

Threshold – The lowest dose that will produce a given effect. As a practical matter, the threshold is little different from the **NOAEL.**

Toxicant – A toxic agent; a poison.

Toxicity – The whole pattern of harmful effects (illness and other undesirable effects) that a chemical can cause. It is a property of the chemical; it does not change.

Toxicology – The group of scientific disciplines that identifies and studies the adverse effects of chemicals on biological systems, whether in the laboratory or in the field.

Tumour – a new growth of cells multiplying progressively and without control. Classically, the term means a swelling.

Titles in this Series

1 Principles of health effects evaluation and risk estimation for chemicals that may be encountered in forest vegetation management

2 Pesticide testing for registration: toxicity, environmental behaviour, and epidemiology

3 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part I: Risk to workers associated with exposure to emissions from power saws

4 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part II: Exposure to and absorption of herbicides used in forestry

5 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part III: Risk to workers using 2,4-D formulations

6 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part IV: Risk to workers using glyphosate formulations (e.g., Vision®, Roundup®, Vantage Forestry® and Forza®)

7 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part V: Risk to workers using hexazinone formulations (Pronone®, Velpar® L)

8 Toxicology and potential health risk of chemicals that may be encountered by forest vegetation management workers. Part VI: Risk to workers using triclopyr formulations (Release®, or Garlon 4®)

9 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options: Summary

Title Number

3

For more information contact:
Dr. Jacob O. Boateng
Vegetation Management Specialist
BC Ministry of Forests
Forest Practices Branch
P.O. Box 9513 Stn. Prov. Govt
Victoria, BC V8W 9C2
Phone: (250) 387-8905
Fax: (250) 387-2136

Titles in this Series

1 Principles of health effects evaluation and risk estimation for chemicals that may be encountered in forest vegetation management

2 Pesticide testing for registration: toxicity, environmental behaviour, and epidemiology

3 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part I: Risk to workers associated with exposure to emissions from power saws

4 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part II: Exposure to and absorption of herbicides used in forestry

5 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part III: Risk to workers using 2,4-D formulations

6 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part IV: Risk to workers using glyphosate formulations (e.g., Vision®, Roundup®, Vantage Forestry® and Forza®)

7 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options. Part V: Risk to workers using hexazinone formulations (Pronone®, Velpar® L)

8 Toxicology and potential health risk of chemicals that may be encountered by forest vegetation management workers. Part VI: Risk to workers using triclopyr formulations (Release®, or Garlon 4®)

9 Toxicology and potential health risk of chemicals that may be encountered by workers using forest vegetation management options: Summary

Title Number

3

For more information contact:
Dr. Jacob O. Bosteng
Vegetation Management Specialist
BC Ministry of Forests
Forest Practices Branch
P.O. Box 9513 Stn. Prov. Govt
Victoria, BC V8W 9C2
Phone: (250) 387-8905
Fax: (250) 387-2136

BRITISH COLUMBIA